=> d his

(FILE 'HOME' ENTERED AT 16:32:10 ON 05 MAY 2006)

FILE 'CAPLUS, MEDLINE, BIOSIS' ENTERED AT 16:32:51 ON 05 MAY 2006

```
L1         10515 S INTERFERON (1W) TYPE (1W) I
L2       1770283 S ANTIBODY
L3            57 S L1 (L) L2
L4             4 S L3 AND ADMINISTRATION
L5            18 S L3 AND TREATMENT
L6            11 DUP REM L5 (7 DUPLICATES REMOVED)
                 E BANCHEREAU JACQUES /AU
L7           569 S E3
                 E PALUCKA ANNA /AU
                 E BLANCO PATRICK /AU
L8            53 S E3
L9           604 S L7 OR L8
L10       157333 S L9 AND INTERFERON OR IFN
L11           93 S L9 AND INTERFERON
L12           60 S L11 AND IFN
L13           19 S L12 AND ANTIBODY
L14           11 DUP REM L13 (8 DUPLICATES REMOVED)
L15            1 S L14 AND TREATMENT
```

L15 ANSWER 1 OF 1 MEDLINE on STN
TI **IFN**-alpha induces early lethal lupus in preautoimmune (New Zealand Black x New Zealand White) F1 but not in BALB/c mice.
AU Mathian Alexis; Weinberg Arthur; Gallegos Mike; **Banchereau Jacques**; Koutouzov Sophie
SO Journal of immunology (Baltimore, Md. : 1950), (2005 Mar 1) Vol. 174, No. 5, pp. 2499-506.
Journal code: 2985117R. ISSN: 0022-1767.
PY 2005
TI **IFN**-alpha induces early lethal lupus in preautoimmune (New Zealand Black x New Zealand White) F1 but not in BALB/c mice.
AU Mathian Alexis; Weinberg Arthur; Gallegos Mike; **Banchereau Jacques**; Koutouzov Sophie
AB Recent studies indicate that **IFN**-alpha is involved in pathogenesis of systemic lupus erythematosus. However, direct proof that **IFN**-alpha is not only necessary, but also sufficient to induce lupus pathogenicity is lacking. In this study, we show that in vivo adenovector-mediated delivery of murine **IFN**-alpha results in preautoimmune (New Zealand Black (NZB) x New Zealand White (NZW))F(1), but not in normal, mice, in a rapid. . . severe disease with all characteristics of systemic lupus erythematosus. Anti-dsDNA Abs appeared as soon as day 10 after initiation of **IFN**-alpha **treatment**. Proteinuria and death caused by glomerulonephritis occurred in all treated mice within, respectively, approximately 9 and approximately 18 wk, at a time when all untreated (NZB x NZW)F(1) did not show any sign of disease. **IFN**-alpha in vivo induced an overexpression of B lymphocyte stimulator in circulation at similar levels in both the preautoimmune and the normal mouse strains. All effects elicited by **IFN**-alpha were dose dependent. (NZB x NZW)F(1) infused with purified murine **IFN**-alpha also showed acceleration of lupus. Thus, prolonged expression of **IFN**-alpha in vivo induces early lethal lupus in susceptible animals.
CT Check Tags: Female; Male
Animals
Antibodies, Antinuclear: BI, biosynthesis
Comparative Study
*Crosses, Genetic
DNA: IM, immunology
Dose-Response Relationship, Immunologic
Genetic Predisposition to Disease
Glomerulonephritis: GE, genetics
Glomerulonephritis: IM, immunology
Glomerulonephritis: MO, mortality
Immunoglobulin G: BI, biosynthesis
Injections, Intravenous
***Interferon-alpha: AD, administration & dosage**
Interferon-alpha: IP, isolation & purification
Lupus Erythematosus, Systemic: GE, genetics
*Lupus Erythematosus, Systemic: IM, immunology
*Lupus Erythematosus, Systemic: MO, mortality
. . .
CN 0 (**Antibodies**, Antinuclear); 0 (B cell activating factor); 0 (Immunoglobulin G); 0 (**Interferon**-alpha); 0 (Membrane Proteins); 0 (Tumor Necrosis Factor-alpha); 0 (**interferon** alpha5)

=> d l11 1-11 ti au py so kwic

L11 ANSWER 1 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI Upon viral exposure, myeloid and plasmacytoid dendritic cells produce 3 waves of distinct chemokines to recruit immune effectors
AU Piqueras, Bernard; Connolly, John; Freitas, Heidi; Palucka, Anna Karolina; **Banchereau, Jacques**
PY 2006
SO Blood (2006), 107(7), 2613-2618
CODEN: BLOOAW; ISSN: 0006-4971
AU Piqueras, Bernard; Connolly, John; Freitas, Heidi; Palucka, Anna Karolina; **Banchereau, Jacques**
IT Chemokines

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Mig (monokine induced by **interferon**-γ); chemokine production by dendritic cells in response to influenza virus infection)

IT Chemokines
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**interferon** γ-inducible protein-10; chemokine production by dendritic cells in response to influenza virus infection)

L11 ANSWER 2 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI Immune and Clinical Outcomes in Patients with Stage IV Melanoma Vaccinated with Peptide-Pulsed Dendritic Cells Derived From CD34+ Progenitors and Activated with Type I **Interferon**
AU **Banchereau, Jacques**; Ueno, Hideki; Dhodapkar, Madhav; Connolly, John; Finholt, Jennifer P.; Klechevsky, Eynav; Blanck, Jean-Philippe; Johnston, Dennis A.; Palucka, A. Karolina; Fay, Joseph
PY 2005
SO Journal of Immunotherapy (2005), 28(5), 505-516
CODEN: JOIMF8; ISSN: 1524-9557
TI . . . in Patients with Stage IV Melanoma Vaccinated with Peptide-Pulsed Dendritic Cells Derived From CD34+ Progenitors and Activated with Type I **Interferon**
AU **Banchereau, Jacques**; Ueno, Hideki; Dhodapkar, Madhav; Connolly, John; Finholt, Jennifer P.; Klechevsky, Eynav; Blanck, Jean-Philippe; Johnston, Dennis A.; Palucka, A. Karolina;. . .

L11 ANSWER 3 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI Spontaneous proliferation and type 2 cytokine secretion by CD4+T cells in patients with metastatic melanoma vaccinated with antigen-pulsed dendritic cells
AU Palucka, A. Karolina; Connolly, John; Ueno, Hideki; Kohl, John; Paczesny, Sophie; Dhodapkar, Madhav; Fay, Joseph; **Banchereau, Jacques**
PY 2005
SO Journal of Clinical Immunology (2005), 25(3), 288-295
CODEN: JCIMDO; ISSN: 0271-9142
AU Palucka, A. Karolina; Connolly, John; Ueno, Hideki; Kohl, John; Paczesny, Sophie; Dhodapkar, Madhav; Fay, Joseph; **Banchereau, Jacques**
IT Chemokines
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**interferon** γ-inducible protein-10; spontaneous proliferation and type 2 cytokine secretion by cd4+t cells in patients with metastatic melanoma vaccinated with antigen-pulsed dendritic cells)
IT **Interferons**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(γ; spontaneous proliferation and type 2 cytokine secretion by cd4+t cells in patients with metastatic melanoma vaccinated with antigen-pulsed dendritic cells)

L11 ANSWER 4 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI Cross-regulation of TNF and IFN-α in autoimmune diseases
AU Palucka, A. Karolina; Blanck, Jean-Philippe; Bennett, Lynda; Pascual, Virginia; **Banchereau, Jacques**
PY 2005
SO Proceedings of the National Academy of Sciences of the United States of America (2005), 102(9), 3372-3377
CODEN: PNASA6; ISSN: 0027-8424
AU Palucka, A. Karolina; Blanck, Jean-Philippe; Bennett, Lynda; Pascual, Virginia; **Banchereau, Jacques**
ST TNF **interferon** alpha autoimmunity lupus arthritis immunotherapy
IT **Interferons**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(α; TNF in expression of and IFN-α regulated genes in therapy of autoimmune diseases)

L11 ANSWER 5 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI IFN-α Induces Early Lethal Lupus in Preautoimmune (New Zealand Black x New Zealand White)F1 but Not in BALB/c Mice
AU Mathian, Alexis; Weinberg, Arthur; Gallegos, Mike; **Banchereau, Jacques**; Koutouzov, Sophie

PY 2005
SO Journal of Immunology (2005), 174(5), 2499-2506
CODEN: JOIMA3; ISSN: 0022-1767
AU Mathian, Alexis; Weinberg, Arthur; Gallegos, Mike; **Banchereau, Jacques**; Koutouzov, Sophie
AB Recent studies indicate that **interferon** α (IFN-α) is involved in pathogenesis of systemic lupus erythematosus. However, direct proof that IFN-α is not only necessary, but. . .
ST **interferon** alpha early lethal lupus susceptible mouse
IT Susceptibility (genetic)
(**interferon** α prolonged expression induces early lethal lupus in susceptible mice)
IT Inflammation
Kidney, disease
(lupus nephritis; **interferon** α prolonged expression induces early lethal lupus in susceptible mice)
IT Lupus erythematosus
(systemic; **interferon** α prolonged expression induces early lethal lupus in susceptible mice)
IT **Interferons**
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(α; **interferon** α prolonged expression induces early lethal lupus in susceptible mice)

L11 ANSWER 6 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI Dendritic cells control B cell growth and differentiation
AU Jego, Gaetan; Pascual, Virginia; Palucka, A. Karolina; **Banchereau, Jacques**
PY 2005
SO Current Directions in Autoimmunity (2005), 8(B Cell Trophic Factors and B Cell Antagonism in Autoimmune Disease), 124-139
CODEN: CDAUF8
AU Jego, Gaetan; Pascual, Virginia; Palucka, A. Karolina; **Banchereau, Jacques**
AB . . . fashion, B cell growth and differentiation. Plasmacytoid DCs drive memory B cell differentiation into effector plasma cell via type I **interferon** and IL-6. Type I **interferon** activates myeloid DCs that regulate B cell priming and acquisition of memory phenotype via IL-12, IL-6 and BLyS/BAFF. This model. . .
ST review dendritic cell B cell differentiation **interferon** interleukin 6; differentiation B cell interleukin 12 BLyS review
IT **Interferons**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type I; cytokines in dendritic cell control of B-cell growth and differentiation)

L11 ANSWER 7 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI Expansion of melanoma-specific cytolytic CD8+ T cell precursors in patients with metastatic melanoma vaccinated with CD34+ progenitor-derived dendritic cells
AU Paczesny, Sophie; **Banchereau, Jacques**; Wittkowski, Knut M.; Saracino, Giovanna; Fay, Joseph; Palucka, A. Karolina
PY 2004
SO Journal of Experimental Medicine (2004), 199(11), 1503-1511
CODEN: JEMEAV; ISSN: 0022-1007
AU Paczesny, Sophie; **Banchereau, Jacques**; Wittkowski, Knut M.; Saracino, Giovanna; Fay, Joseph; Palucka, A. Karolina
AB . . . histocompatibility leukocyte antigen (HLA)-A*0201 patients with metastatic melanoma, that vaccination with peptide-loaded CD34-dendritic cells (DCs) leads to expansion of melanoma-specific **interferon** γ-producing CD8+ T cells in the blood. Here, the authors show in 9 out of 12 analyzed patients the expansion. . .

L11 ANSWER 8 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI Dendritic cells loaded with killed breast cancer cells induce differentiation of tumor-specific cytotoxic T lymphocytes
AU Neidhardt-Berard, Eve-Marie; Berard, Frederic; **Banchereau, Jacques**; Palucka, A. Karolina
PY 2004

SO Breast Cancer Research (2004), 6(4), R322-R328
CODEN: BRCRFS; ISSN: 1465-542X
URL: http://breast-cancer-research.com/content/pdf/bcr794.pdf
AU Neidhardt-Berard, Eve-Marie; Berard, Frederic; **Banchereau, Jacques**; Palucka, A. Karolina
AB . . . and CD4+ T lymphocytes. The elicited CTLs are able to kill the target cells without a need for pretreatment with **interferon** gamma. CTLs can be obtained by culturing the DCs loaded with killed breast cancer cells with unsepd. peripheral blood lymphocytes,. . .

L11 ANSWER 9 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI Autoimmunity through cytokine-induced dendritic cell activation
AU **Banchereau, Jacques**; Pascual, Virginia; Palucka, A. Karolina
PY 2004
SO Immunity (2004), 20(5), 539-550
CODEN: IUNIEH; ISSN: 1074-7613
AU **Banchereau, Jacques**; Pascual, Virginia; Palucka, A. Karolina
AB . . . autoimmune responses. When balanced, both cytokines synergize in protective immunity. When one of the cytokines prevails, autoimmunity occurs, Type I **interferons** (IFN-α/β) playing a major role in systemic lupus erythematosus (SLE) and TNF playing a major role in rheumatoid arthritis. This. . .
ST review autoimmunity dendritic cell **interferon** interleukin SLE
IT **Interferons**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(α; cytokine-induced dendritic cell activation in autoimmunity)
IT **Interferons**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(β; cytokine-induced dendritic cell activation in autoimmunity)
IT **Interferons**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(γ; cytokine-induced dendritic cell activation in autoimmunity)

L11 ANSWER 10 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI Dendritic cells generated in the presence of GM-CSF plus IL-15 prime potent CD8+ Tc1 responses in vivo
AU Pulendran, Bali; Dillon, Stephanie; Joseph, Chryshanthi; Curiel, Tyler; **Banchereau, Jacques**; Mohamadzadeh, Mansour
PY 2004
SO European Journal of Immunology (2004), 34(1), 66-73
CODEN: EJIMAF; ISSN: 0014-2980
AU Pulendran, Bali; Dillon, Stephanie; Joseph, Chryshanthi; Curiel, Tyler; **Banchereau, Jacques**; Mohamadzadeh, Mansour
IT **Interferons**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(γ; dendritic cells generated in presence of GM-CSF plus IL-15 prime potent CD8+ Tc1 responses in vivo)

L11 ANSWER 11 OF 93 CAPLUS COPYRIGHT 2006 ACS on STN
TI Dendritic cells: controllers of the immune system and a new promise for immunotherapy
AU **Banchereau, Jacques**; Fay, Joseph; Pascual, Virginia; Palucka, A. Karolina
PY 2003
SO Novartis Foundation Symposium (2003), 252(Generation and Effector Functions of Regulatory Lymphocytes), 226-238
CODEN: NFSYF7; ISSN: 1528-2511
AU **Banchereau, Jacques**; Fay, Joseph; Pascual, Virginia; Palucka, A. Karolina
ST review dendritic cell immunoregulation immunotherapy **interferon** melanoma vaccine autoimmunity
IT **Interferons**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(α; dendritic cells in immunoregulation and immunotherapy of tumor and autoimmune diseases)

```
=> d his

     (FILE 'HOME' ENTERED AT 16:32:10 ON 05 MAY 2006)

     FILE 'CAPLUS, MEDLINE, BIOSIS' ENTERED AT 16:32:51 ON 05 MAY 2006
L1         10515 S INTERFERON (1W) TYPE (1W) I
L2       1770283 S ANTIBODY
L3            57 S L1 (L) L2
L4             4 S L3 AND ADMINISTRATION
L5            18 S L3 AND TREATMENT
L6            11 DUP REM L5 (7 DUPLICATES REMOVED)
```

L6 ANSWER 1 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
TI Type I interferon blocking agents for prevention and **treatment** of psoriasis
IN Gilliet, Michel; Nestle, Frank O.
SO PCT Int. Appl., 20 pp.
CODEN: PIXXD2
PY 2006
TI Type I interferon blocking agents for prevention and **treatment** of psoriasis
AB . . . (e.g. an anti-IFN-α antibody) or type I IFN receptor antagonist, for the preparation of a medicament for the prevention and **treatment** of psoriasis, and to a method of prevention and **treatment** of psoriasis using a type I interferon blocking agent.
ST psoriasis **interferon type I** blocker **antibody**
IT Fusion proteins (chimeric proteins)
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(IFN/FC; type-I interferon blocking agents for prevention and **treatment** of psoriasis)
IT Antibodies and Immunoglobulins
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(anti-IFN-α; type-I interferon blocking agents for prevention and **treatment** of psoriasis)
IT Antibodies and Immunoglobulins
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(fragments, anti-IFN-α; type-I interferon blocking agents for prevention and **treatment** of psoriasis)
IT Antibodies and Immunoglobulins
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(humanized, anti-IFN-α; type-I interferon blocking agents for prevention and **treatment** of psoriasis)
IT Double stranded RNA
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(small interfering; type-I interferon blocking agents for prevention and **treatment** of psoriasis)
IT Interferons
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type I, antagonists; type-I interferon blocking agents for prevention and **treatment** of psoriasis)
IT PCR (polymerase chain reaction)
Psoriasis
Signal transduction, biological
(type-I interferon blocking agents for prevention and **treatment** of psoriasis)
IT Interferon receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type-I interferon blocking agents for prevention and **treatment** of psoriasis)
IT Antisense oligonucleotides
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(type-I interferon blocking agents for prevention and **treatment** of psoriasis)
IT Interferons
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(α, antagonists; type-I interferon blocking agents for prevention and **treatment** of psoriasis)
IT 882706-06-1 882706-07-2 882706-08-3 882706-09-4
RL: PRP (Properties)
(unclaimed nucleotide sequence; type I interferon blocking agents for prevention and **treatment** of psoriasis)

L6 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
TI Interleukin-12- and gamma interferon-dependent innate immunity are essential and sufficient for long-term survival of passively immunized mice infected with herpes simplex virus type 1
AU Vollstedt, Sabine; Franchini, Marco; Alber, Gottfried; Ackermann, Mathias; Suter, Mark
SO Journal of Virology (2001), 75(20), 9596-9600
CODEN: JOVIAM; ISSN: 0022-538X
PY 2001
AB **Interferon** (IFN) **type I** (α/β IFN) is very important in directly controlling herpes simplex virus type 1 (HSV-1) replication as well as in guiding and upregulating specific immunity against this virus. By contrast, the roles of IFN type II (IFN-γ) and **antibodies** in the defense against HSV-1 are not clear. Mice without a functional IFN system and no mature B and T cells (AGR mice) did not survive HSV-1 infection in the presence or absence of neutralizing **antibodies** to the virus. Mice without a functional IFN type I system and with no mature B and T cells (AR129. . . with as little as 10 PFU of HSV-1 strain F. By contrast, in the presence of passively administered neutralizing murine **antibodies** to HSV-1, some AR129 mice survived infection with up to 104 PFU of HSV-1. This acute immune response was dependent on the presence of interleukin-12 (IL-12) p75. Interestingly, some virus-infected mice stayed healthy for several months, at which time **antibody** to HSV-1 was no longer detectable. **Treatment** of these virus-exposed mice with dexamethasone led to death in approx. 40% of the mice. HSV-1 was found in brains of mice that did not survive dexamethasone **treatment**, whereas HSV-1 was absent in those that survived the **treatment**. Thus, in the presence of passively administered HSV-1-specific **antibodies**, the IL-12-induced IFN-γ-dependent innate immune response is able to control low doses of virus infection. Surprisingly, in a proportion of these mice, HSV-1 appears to persist in the absence of **antibodies** and specific immunity.

L6 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
TI Resistance to influenza virus infection of Mx transgenic mice expressing Mx protein under the control of two constitutive promoters
AU Kolb, E.; Laine, E.; Strehler, D.; Staeheli, P.
SO Journal of Virology (1992), 66(3), 1709-16
CODEN: JOVIAM; ISSN: 0022-538X
PY 1992
AB . . . virus strain NWS. Control mice of the A2G strain express Mx1 protein in all organs, but only after induction by **interferon type I** upon or without viral infection. The extent of specific resistance in transgenic mice of the best-expressing line reached about 2/3. . . virus agent and that its efficiency in the exptl. setting is independent of interferon actions could be derived from the **treatment** of exptl. and control mice with anti-interferon **antibodies** at the time of virus tests. Whereas in A2G mice, Mx1 mRNA and Mx1 protein synthesis were abolished and viral. . .

L6 ANSWER 8 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI THERAPEUTICAL TRIAL WITH INTERFERON IN SUBACUTE SCLEROSING PANENCEPHALITIS SSPE.
AU FELDMANN M [Reprint author]; SCHAEFER U; PRANGE H W
SO Aktuelle Neurologie, (1989) Vol. 16, No. 3, pp. 93-98.
ISSN: 0302-4350.
PY 1989
AB . . immunity in the host nor for genetic immunodefective factors. In the last few years, there have been numerous trials of **interferon type I** (Ifn-α, -β) in the **treatment** of SSPE, but critical considerations do not reveal any long-term positive effect on the progressive clinical course of the disease. . . sustained clinical improvement either. In our opinion, the transient change in serum or CSF IgG-concentrations and in the measles virus **antibody** titres during interferon application cannot be seen a specific antiviral effect, but rather as a non-specific depression of IgG-producing B-lymphocytes.. . .

L6 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 3
TI Effect of interferon and interferon inducers on the NK sensitivity of normal mouse thymocytes
AU Hansson, Mona; Kiessling, Rolf; Andersson, Birger; Welsh, Raymond M.
SO Journal of Immunology (1980), 125(5), 2225-31
CODEN: JOIMA3; ISSN: 0022-1767
PY 1980
AB . . . contrast, LCMV carrier mice, infected congenitally, had normal levels of thymus NK sensitivity and no NK activation or interferon synthesis. **Treatment** of thymocytes in vitro with **interferon type I** at concns. of >103 units dramatically reduced their NK sensitivity, without concomitant cytotoxicity. For strongest protection, 7-14 h incubation was. . . reversed by an anti-interferon serum. Interferon-treated thymocytes also had increased levels of serol. defined H-2 antigens and increased sensitivity for allo-**antibody**-induced cytotoxicity. By cold target inhibition studies, it was demonstrated that interferon-treated thymocytes expressed less of the NK target structure expressed. . .

L6 ANSWER 10 OF 11 MEDLINE on STN DUPLICATE 4
TI Effect of virus-induced interferon on the antibody response of suckling and adult mice.
AU Vignaux F; Gresser I; Fridman W H
SO European journal of immunology, (1980 Oct) Vol. 10, No. 10, pp. 767-72.
Journal code: 1273201. ISSN: 0014-2980.
PY 1980
AB Continued administration of potent virally induced mouse **interferon** (IFN **type I**) preparations to suckling mice resulted in an inhibition of the primary **antibody** response to sheep erythrocytes (14-day-old mice). When slightly older suckling mice were immunized (17 days old), a delay (about 2. . . the response was similar in both control and IFN-treated animals. In adult mice, potent IFN preparations did not inhibit the **antibody** response under a variety of experimental conditions (different doses of IFN, schedules of **treatment**, routes of injection, times of assay). Although IFN does inhibit the in vitro **antibody** response, we conclude that under most experimental conditions, injection of IFN type I is not immunosuppressive in adult mice. When administered after immunization, IFN clearly enhanced the primary **antibody** response. It is of interest that IFN, a product of viral infection, enhances in vivo those components of the immune. . . delayed-type hypersensitivity, natural killer cell and macrophage activity, expression of lymphocyte membrane molecules), and, as is shown here, the primary **antibody** response.

L6 ANSWER 11 OF 11 MEDLINE on STN DUPLICATE 5
TI Cytotoxic cells induced during lymphocytic choriomeningitis virus infection of mice: natural killer cell activity in cultured spleen leukocytes concomitant with T-cell-dependent immune interferon production.
AU Welsh R M Jr; Doe W F
SO Infection and immunity, (1980 Nov) Vol. 30, No. 2, pp. 473-83.
Journal code: 0246127. ISSN: 0019-9567.
PY 1980
AB . . . adherent peritoneal cells was due to contamination with cytotoxic T cells, as shown by H-2-restricted cytotoxicity and sensitivity to anti-theta **antibody** and complement. The nonspecific cultured day 6 effector cell from either the spleen or peritoneum displayed killing specificities and other physical properties identical to those of activated NK cells, but had sensitivities to anti-theta **antibody** and complement intermediate between activated day 3 NK cells and cytotoxic T cells. Culture stable NK-like cells were not found. . . Significant levels of interferon were produced by nylon-wool-passed day 6 spleen cells, and virtually all interferon production was eliminated by **treatment** of either day 2 or day 6 cells with **antibody** to theta antigen and complement, suggesting that T cells produced the interferon in vitro. Furthermore, athymic nude mice had number . . cells 6 days postinfection, and spleen cells from them failed to produce significant levels of interferon in vitro. Addition of **interferon**

(type I, fibroblast) to cultured C3H spleen cells affect the already elevated levels of cytotoxicity in day 6 cultures, suggesting that the. . .